steadily; in two weeks foot-drop disappeared. After three weeks fine movements of hands became possible and anaesthesia was absent. Patient was discharged after six weeks; shortly beforehand paraesthesiae had gone, but although power greatly improved, it would not permit severe exertion.

Severe Cases

Of the series only three cases became bed-ridden; one of these is described.

Case 12.—Gave a history of jungle sores beginning two months previously; finally healed a few days before admission. Present complaints: tingling of extremities, unsteadiness of gait, and blurring of vision. These occurred at approximately the same time as the sores healed. On examination, general condition good. No evidence of avitaminosis or anaemia. Scars of healed jungle sores on legs and forearms. All systems normal except C.N.S. C.N.S. examination: pupils and cranial nerves normal; weakness of grip—L. > R.; slight weakness of extensors of wrist and dorsiflexors of ankle; no anaesthesia; upper-limb tendon reflexes present; ankle- and knee-jerks absent on both sides. Two days after admission:—Tendency to foot-drop, L.>R.; impairment of all forms of sensation up to knees; astereognosis present; biceps jerks now absent. There followed a rapid increase in weakness, maximal peripherally and spreading towards the trunk. Ten days after admission:—Patient unable to move either arms or legs; muscles of both hips and shoulders weak; sensation bilaterally impaired up to the elbows, and on legs up to and including the perineum; no disturbance of sphincters; all tendon reflexes absent; cremasteric and abdominal reflexes present. One month after admission:—First signs of recovery appeared; power of hips and shoulders began to return. Six weeks after admission:—Able to lift arms above head; power returning to upper arms and thighs; foot-drop improved; area of anaesthesia greatly diminished. Eight weeks after admission:—Able to use hands and stand with support; extremities still weak and with impaired sensation; all tendon reflexes absent. The paraesthesiae, which had disappeared at the time of maximum anaesthesia and had returned in the early stages of recovery, now left him entirely. At this point the patient was transferred to a rehabilitation centre.

Discussion

There is no doubt that these patients were suffering from a polyneuritis, the aetiology of the condition being the main point of interest. Chemical poisons such as arsenic can be excluded as the aetiological agent, since no patient gave a history of exposure to this. Acute infective polyneuritis is excluded on the natural history of the disease, the onset in the series being insidious and the course afebrile. It remains to decide whether the polyneuritis is the result of a vitamin deficiency or is due to a bacterial toxin. Cases of vitamin B deficiency were seen in troops engaged in the same operation; the picture presented by these was primarily one of wasting and diarrhoea, with glossitis. Polyneuritis, when present, was mild and responded quickly to intensive vitamin therapy.

In the cases under discussion the polyneuritis, as has been shown, could be completely disabling, and was not influenced by adequate doses of the vitamin B complex. Moreover, the nutrition of all cases was good, despite the conditions under which they had been living, no glossitis and no diarrhoea being noticed.

Since a constant association between jungle sores and a subsequent polyneuritis was noticed, it is likely that a toxin, elaborated by bacteria infecting these ulcers, was the responsible agent. Clinically a marked similarity exists between jungle sores and the desert sores seen in troops of the M.E.F. It was found that a number of desert sores were infected with diphtheria bacilli; similarly these organisms have been isolated from jungle sores. In our series of polyneuritic cases, except for one patient, the sores were healed at the time of admission. From the unhealed sores of this case (No. 19) diphtheria bacilli were isolated. However, a week after admission this patient developed faucial diphtheria; therefore it is difficult to decide whether the polyneuritis was due to the faucial or to the extrafaucial infection.

The time of onset of polyneuritis indicated the latter. The outstanding clinical point in favour of a diphtheritic aetiology is the paralysis of accommodation noted in a large percentage of cases. It is also important to note that the neuritis was not locally related to those areas affected by jungle sores. In a few cases neuritis of the legs and arms followed when the ulcers had been present only on the arms, and vice versa. The loss of accommodation can also be explained by assuming a blood-borne spread of the toxin. This form of generalized peripheral neuritis is seen in both faucial and extrafaucial diphtheria, and as a clincali entity differs from other types of polyneuritis. The number of ulcers did not influence the severity of the disease; similarly, neither did the duration of unhealed jungle sores. A feature of interest was the fact that although Indian troops taking part in the same operation developed jungle sores, no case of a subsequent polyneuritis was discovered. It is well known that diphtheritic infection is rare in Indians.

From the above facts it is felt that all the cases represent a polyneuritis of diphtheritic origin. The value of Schick testing was obvious, but although every attempt was made to obtain the toxin none was available. Electrocardiographic studies would also have been valuable. The origin of a diphtheritic infection incurred in the jungle is obscure. These cases of polyneuritis were drawn from all the units concerned in the operation. If a carrier was the source of infection it is likely that the cases would have been grouped in one unit. All troops came into contact with the native population, and it is more likely that the infection was introduced from them. However, no details are known of the incidence or carrier rate of diphtheria in the Burmese.

From a military point of view this disease can cause a serious wastage of man-power. Even the mild cases will be unfit for jungle warfare for six months from the onset of their disability. The severe cases will probably take a year to recover fully. These men neither had been immunized against diphtheria nor had previously suffered from the disease. If the diphtheritic origin of this polyneuritis is accepted the question of prophylactic immunization will arise for troops engaged in future jungle operations.

Summary

Twenty-one cases of polyneuritis subsequent to jungle sores are described.

The course of the disease followed a constant pattern.

Sixteen out of 21 cases had loss of accommodation.

In reviewing the aetiology it was considered most likely that the polyneuritis followed a diphtheritic infection of the jungle sores.

We wish to thank Col. Wm. Morrison, *M.C.*, F.R.C.S.Ed., officer commanding a general hospital, for his advice and friendly criticism, and also for permission to publish this article.

ACUTE INFECTIVE POLYNEURITIS

BY

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A form of acute polyneuritis apparently of infective origin is well recognized by neurologists but has received little attention in English medical textbooks. It is probably more common than polyneuritis due to the classical toxins, and seems often to be a source of diagnostic confusion. The following case, which appears to be typical, seems therefore worth recording.

Case Report

An air-gunner aged 23 was admitted to his station sick-quarters with a left facial palsy. On the day of admission he rapidly developed " pins and needles " in arms and legs, with marked muscular weakness. The condition was recognized as a generalized one, and he was transferred to hospital. There was no sphincter disturbance. On admission to hospital it was elicited that he had had a bad cold and chill a fortnight previously. Apart from appendicectomy a year ago, he had always been healthy and never a heavy drinker. On examination he was afebrile, but appeared ill. A complete left facial palsy of the lower motor neurone type was present, but the other cranial nerves were normal. The fundus oculi was healthy. There was marked muscular weakness of arms and legs, with winging of the right scapula. The proximal muscle groups, indeed, were particularly affected. Tendon reflexes could still be obtained on admission, but within a few days had entirely disappeared from the legs and left arm, and were feeble in the right arm. The muscles were flaccid and toneless. The upper abdominal reflexes were present, but not the lower; the plantars were flexor. No reliable sensory changes could be discovered, but the paraesthesiae were the cause of much complaint. The throat was clear, the nose a little congested. No K.L.B. could be isolated from a swab. Other systems were normal. Lumbar puncture on the day after admission showed a clear fluid under normal pressure, with no cells; protein,

45 mg. per 100 c.cm.; Wassermann reaction negative. Recovery from the facial palsy was rapid, being complete within a month. In the arms it was slower, but tendon reflexes were present in three weeks and normal in four. The legs were slowest of all, and convalescence was attended by much aching in the feet. After nine weeks the lower-limb reflexes could be obtained, with the exception of the left ankle-jerk. By 11 weeks he was walking well with the aid of a stick, and shortly afterwards was transferred for rehabilitation. All tendon reflexes had returned by this time, but there was residual weakness of the glutei and calf muscles, with still a little pain in the feet.

Discussion

A description of this condition, with post-mortem findings, was given by Gordon Holmes (1917) under Osler's diagnosis of acute febrile polyneuritis. A more detailed account, based on 30 cases, was published by Bradford, Bashford, and Wilson (1919). Transmission of the disease to monkeys from post-mortem material is described and its infectious origin held to be established. Hence the term "acute infective polyneuritis," which has since been commonly used. In the present war a group of 7 cases has been described by Barber (1940) under the title "Polyradiculoneuritis (Guillain-Barré Syndrome)," which he gives reasons for considering the same as Holmes's. From the descriptions this would appear reasonable, though Guillain (1936), writing 20 years after his original publication, still wished to draw a distinction between the syndrome known by his name and the type characterized by fever. The eponym, much used by French writers, pays tribute to his work on the subject. American authors have added other series in recent years. Forster, Brown, and Merritt (1941) describe 26 similar cases as "polyneuritis with facial diplegia," although facial involvement was not a constant feature, being lacking in four patients. They point out incidentally that their cases showed a much worse prognosis than is usually given, 11 ending fatally, and suggest that an increase in virulence of the hypothetical causative virus occurred at one period of their survey. An interesting possibility that has often been discussed is that Landry's paralysis is a form of the disease under consideration (Pinckney, 1936; Garvey and Slavin, 1938). A long list of synonyms is given in the recent editions of Osler's *Medicine*.

The importance of establishing the diagnosis is not entirely academic, in spite of our ignorance of the aetiology and the specific therapy. Cases are often labelled anterior poliomyelitis, and a much more serious prognosis given as regards paralysis than is warranted. On the other hand, failure to appreciate the possibility of involvement of the respiratory muscles may assist in fatal consequences. Points of differential diagnosis in poliomyelitis are given by Gordon Holmes as "the more rapid evolution and the more irregular distribution of the palsy, the frequent paralysis of the trunk muscles, and the usual absence of objective sensory symptoms in this disease." It is also generally agreed that in the polyneuritis there is usually a considerable interval between the febrile episode, which may mark the onset of the illness, and the appearance of the characteristic symptoms. Recovery, too, tends to be complete. The C.S.F. findings show in polyneuritis an increase of cells—an acellular albuminosis, as it has been called. This was not marked in the present case, but the sample was obtained very early. I have usually noted higher figures for protein in civilian cases, which come to hospital at a later stage of the disease. On the other hand, seven of Forster, Brown, and Merritt's cases had protein within normal limits, and they find that this cannot be explained on a time basis.

This case was apparently a sporadic one, and no evidence of a local epidemic has been found.

My thanks are due to the Air Ministry for permission to submit this note.

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S. J. Carter (*Ann. Surg.*, 1945, **122**, 117), who records eleven illustrative cases, maintains that serum amylase determination should be made in all chronic alcoholic patients if operation is contemplated for severe acute abdominal symptoms.

Medical Memoranda

Misleading Evidence in Establishing the Time of Death

The rate of emptying of the stomach is considered a useful if subsidiary means of establishing the time of death in medico-legal cases. The following case presents unusual features in this connexion.

Case Record

The dead body of a soldier aged 26 was admitted to the mortuary of a military hospital in the M.E.F. He had sustained severe head injuries when the truck which he was driving overturned, and he died 13 minutes later without recovering consciousness. The body was placed in the mortuary refrigerator 4 hours after death, and the necropsy was performed 14 hours later.

The face and forehead were gashed and abraded, and the left maxilla driven backwards, with depression of the floor of the left orbit, indicating a severe blow on the face. The skull was widely comminuted and the undersurface of the brain lacerated. On section, the mid-brain was peppered with small perivascular haemorrhages and the ventricular fluid was heavily blood-stained. The lungs contained a quantity of blood inhaled from the nasopharynx. A small tear was found in the liver, and the pelvis was fractured.

The stomach was empty; but, lying free in the jejunum, 12 inches below the duodeno-jejunal flexure, was a vulcanite denture with 8 jagged points round the convex border, and bearing one artificial tooth. Its shape was roughly triangular, the base 38 mm., the altitude 25 mm., and it was curved to fit the hard palate. Passage of the denture appeared to have caused no injury to the food channels.

Comment

Subsequent interrogation of a companion in the truck disposed of the idea that the deceased had swallowed his denture before the accident, and it is reasonably certain that it was dislodged at the time of the facial injury. It would appear that the denture had then been swallowed while the patient was unconscious, and passed through the oesophagus, stomach, and duodenum into the jejunum, travelling 45 inches from its original position in the mouth in the course of 13 minutes.

Sydney Smith in his *Forensic Medicine* states that after a full meal food begins to leave the stomach in 15 minutes, and that the stomach is empty in 2 to 5 hours; that a barium meal passes through the stomach almost at once, but that metallic foreign bodies may be retained in the stomach for days; and that food travels through the small intestine at the rate of about 6 feet in an hour.

The surprising distance travelled by the denture may have been due to accelerated peristalsis during life or persistent peristalsis after death; but, in either case, had the time of death been unknown the evidence of the denture would have proved misleading.

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Scabies and D.D.T.

The success of D.D.T. in preventing infection by lice has caused many people to wonder whether it has any effect on scabies. There is little information about this. Buxton (1945) in his excellent account of D.D.T. does not mention scabies, but in the discussion of his paper K. Mellanby says—"D.D.T. is certainly not to be recommended for the treatment of scabies." To determine whether D.D.T. has any prophylactic action against scabies, I examined, in conjunction with Major G. A. Hodgson, a series of infected soldiers to see how many had been wearing impregnated shirts or had been using D.D.T. powder regularly and recently; "recently" was interpreted as being within the last two months. We obtained the following results:

	Scabies Patients			Controls		
	Cases	Yes	No	Cases	Yes	No
G. A. H. ..	48	23 (48%)	25 (52%)	111	46 (41%)	65 (59%)
F. F. H. ..	52	29 (56%)	23 (44%)	100	64	36
Total ..	100	52%	48%	211	110 (52·2%)	101 (47·8%)

("Yes" signifies wearing an impregnated shirt and/or having used D.D.T. powder regularly within the last two months.)

The difference between the proportions protected in the two series is due to their having been observed, purposely, in two different areas. The almost exact identity between the scabies patients and the controls shows that D.D.T. has no preventive action against the acquirement of scabies.

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